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VASCULAR INJURIES DUE TO COLD WITH PARTICULAR REFERENCE TO THE LATE PHASE OF TRENCH FOOT

Cold produces intracellular damage, which may be irreversible. The injury is roughly proportional to the intensity of the cold and to the duration of exposure. The distal portions of the extremities are particularly vulnerable because they are frequently exposed, poorly insulated and susceptible to trauma and because the peripheral circulation with its heat-regulating function is quick to reflect changes in the environmental temperature.

Types of cold trauma

Although the various types of cold injury have distinctive labels which make them appear to have diverse origins, they actually have much in common.

In the extreme cold of the stratosphere, the tissues of a hand which becomes exposed through loss of a glove may be frozen solid almost instantaneously.

The "ground type" of frost-bite follows exposure of a limb for a few hours to subzero temperatures, resulting in partial or complete freezing of tissues.

Prolonged exposure to cold and moisture produces trench foot. This can develop in as short a time as a few days but the period of exposure sometimes runs into weeks. Even when the environmental temperature is above 32° F. a cold injury can occur in a wet extremity through abstraction of heat from the tissues, since water conducts heat better than does air. Other factors, such as immobility, dependency and tight shoes and leggings, aggravate the thermal trauma.

Immersion foot, the clinical counterpart of trench foot, occurs in shipwreck victims who have existed in lifeboats or rafts for varying periods. Starvation, the dependent position of the legs, lack of muscular movements and maceration of the tissues by water play prominent roles in producing the resulting clinical picture.

Finally, in chilblain or pernio there has been exposure of the acral portions of the limbs, especially the pressure or bunion areas or the tip of the nose or lobes of the ears, on repeated occasions. Chronic erythema develops in such localized areas and these areas are painful even when the outside temperature rises to normal levels. In essence, chilblain is a mild type of frostbite of the superficial tissues.

Pathologic physiology

The wealth of clinical material comprising the various thermal injuries in World War II has done much to dispel the obscurity which hitherto has surrounded them. In actual freezing of soft tissues, such as occurs below a critical temperature of —21° F., ice crystals form in the cells, in the walls of the vessels and in the tissue spaces, disrupting the cellular structure and resulting in necrobiosis. Such direct damage may be extreme, resulting in loss of an entire limb or parts thereof. More commonly, however, only the superficial tissues are so involved. Damage to the deeper tissues usually is secondary to change in the blood vessels and this, in reality, is the all-important factor from the viewpoint of viability of the tissues.

Studies in the case of trench foot have yielded fruitful results, which are representative of the changes found in all cold injuries. During the period of exposure there is a constriction of vascular elements of all types and sizes, even of vessels of the caliber of the dorsalis pedis and posterior tibial arteries. This probably represents reflex spasm due to general chilling of the body and is compensatory, to prevent further dissipation of heat. Aschoff's recent experiments on strips of excised artery show that the primary response to cold is one of vasodilatation. Biopsy specimens taken during the ischemic stage show little more than arteriolar constriction; there is no change of cellular structure although it is known that profound changes of cellular metabolism have taken place. Dissociation of hemoglobin is almost at a standstill. During this anoxic phase the capillaries become permeable and there is exudation of fluid into the tissue, producing some swelling. The affected member appears pale or waxy yellow and may remain in this algid state for days or even weeks.

Once thawing sets in, a dramatic change takes place. Cellular metabolites, such as H substance, carbon dioxide and so forth, which have accumulated locally even though activity in the cell has been at a low ebb, result in intense vasodilatation. Grossly, the limb is warm and red or purplish red. Microscopically the vascular endothelium, which has appeared more or less normal until now, becomes swollen and the lumina contain agglutinative thrombi. The muscle fibers in the media of the arterioles are swollen and vacuolated. There is dissolution of cellular boundaries and the edema, which was moderate during the ischemic phase, now increases to high proportions. The escape of fluid and cellular elements into the surrounding tissues results in further embarrassment of the arterial supply. Blebs and blisters form rapidly. There may be rupture of small vessels, resulting in subcutaneous hemorrhage. Necrosis of the skin is due in part to direct injury by cold and secondarily to capillary thrombosis. It is important to realize, as has been so clearly stated by Wilson, that these lesions of soft tissue are not due to the primary injury by cold, but to the "sudden re-establishment of the circulation with its resultant paralytic dilatation of the vessels, intense exudation and circulatory stasis leading to malnutrition." Superimposed pyogenic infection frequently complicates the picture. Fortunately, most patients pass uneventfully into the chronic phase of trench foot without deep necrosis and eventually to complete recovery.

State of the circulation in cases of late trench foot

The symptoms of late trench foot—pain, swelling, sweating, coldness and changes of color—have given rise to the statement that this disease is primarily a circulatory disorder. Some of the investigative work refuting this extreme viewpoint is presented.

The large blood vessels.—Personal examination of more than 1,000 patients and the records of 4,000

more showed the peripheral arteries to be normal clinically; none showed evidence of arterial insufficiency. Bounding pulses frequently were felt in cases in which gangrene of all the toes or even of the distal half of the foot was present. Oscillometric readings simply corroborated the foregoing findings.

Phlebitis of the saphenous vein directly following trench foot was encountered in a few cases. It was surprising to find such a low incidence inasmuch as cold is a frequent predisposing or even exciting factor in producing thrombophletitis in civilian groups. Patterson made the same observation in his study of patients who had cold injuries sustained in the Kiska and Attu campaign.

On several occasions patients who had sharply localized pain, warmth and swelling over the mesial aspect of the ankle near the internal malleolus were seen. It was felt that this condition represented phlebitis of the plantar veins of the foot, although this could not be proved.

The smaller blood vessels. — The smaller blood vessels were studied in a variety of ways.

1. Examination by capillary microscopy of the nailbeds of the fingers and toes of patients who had late trench foot showed nothing which could be considered peculiar to this condition. The arrangement of the capillaries was regular and the corpuscular flow was normal despite clinical evidences of extensive involvement of sensory nerves or in the presence of marked sweating. In patients who had pallor of the hands and feet, the capillaries were narrow but normal in number, arrangement and blood flow; in patients who had marked cyanosis or rubor, the capillary loops exhibited varying degrees of dilatation and slowing of blood flow through them, amounting to complete cessation of blood flow in the extreme cases. In patients with edema, the capillary pattern was similar to those seen in patients who had pale skin. Such findings have been corroborated by Schecter and Ragen in cases of early trench foot encountered in advance echelons.

The one outstanding observation from direct study of the capillaries is that they are not uniformly dilated or paralyzed—vasomotor tone is present—and blood flow through the capillary loop continues with the usual variations. A recent statement by Freedman based on a study of fixed tissue sections in which he described a paralytic dilatation of the capillaries as an essential part of the pathologic changes, probably is applicable only to the very early stages of trench foot.

2. On performance of the test of reactive hyperemia any of the patients who had moderate degrees of trench foot, without open cutaneous lesions, the flushing was seen to reach the distal portion of the extremities, the toes or fingers, in less than five seconds, was most intense in fifteen to twenty seconds and subsided in one to two minutes.

In patients who had gangrene of one or more toes, reactive hyperemia was prompt in appearing (within five seconds in the uninvolved toes and tissue) and prompt in reaching maximal intensity. It was difficult to determine the subsidence of the reaction in this group inasmuch as all had some degree of inflammatory flush surrounding the area of necrosis.

In a small group of patients who had severe degrees of cold injury, characterized by long, persistent and pronounced swelling but without gangrene, reactive hyperemia was delayed, appearing in from seven to twenty-one seconds, reaching its full intensity in thirty to sixty seconds and finally disappearing in five to six minutes instead of in one to two minutes. In still other patients who had marked cyanosis, the flushing was not uniform but blotchy. Such delayed and abnormal responses could be interpreted to indicate either organic change or vasospasm of the smaller vessels, or both. That the blood vessels in the blanched areas are capable of dilatation will be shown later.

Measured by their response to reactive hyperemia, the small vessels are involved clinically in only a small proportion of the cases of late trench foot.

3. On performance of the histamine flare test adequate wheals and flares were found uniformly even in the patients whose circulation appeared impaired during the hyperemia tests. From these studies it is clear that in trench foot the vessels of the skin are organically normal.

4. Study of the tissue circulation by means of the fluorescein test of Lange and Boyd, using a photo-electric cell as an indicator, showed that the circulation time (arm vein to foot) was approximately twenty-two seconds in patients who had trench foot both with and without gangrenous lesions. This is identical with the values obtained in a series of control patients hospitalized for other conditions and studied by the same technic.

5. Studies of cutaneous temperature were not carried out quantitatively. However, mention must be made of feet that are warm but yet cyanotic, a condition not at all rare in the late stages. This incongruous combination can be explained on the basis of an inflammatory reaction due to cold injury in the subcutaneous and deeper tissues which is masked by dilatation and stasis in the cutaneous venous channels, which are responsible for the skin color. That other factors, such as shunting of blood through arteriovenous anastomoses, also may contribute to this state of affairs is a possibility. The surface temperature of the feet in the vast majority of our patients was generally quite cool, owing mainly, in our opinion, to evaporation of sweat.

Comment

The evidence presented shows that the blood vessels in cases of late trench foot for the most part are not organically involved. Nothing conclusive, however, can be said about functional changes which most certainly have to do with skin temperature, cyanosis and flushing. It is our belief, based only on clinical impression, that the small blood vessels are in a sensitized state, responding abnormally in a qualitative and quantitative sense to their usual stimuli. We have been encouraged in this belief by the appearance of a typical Raynaud syndrome in a few cases in which trench foot had developed approximately one year before. The appearance of such syndromes in men more than thirty years of age and in whom such phenomena had not been present previously leads to speculation as to the likelihood that in additional cases of this disorder such symptoms may develop in the next few years.

For the most part, the perpetuation of symptoms in late cold injuries depends on the slow resolution of a chronic sterile inflammatory process with fibrosis within the tissues rather than on organic changes in the blood vessels themselves. These changes have been demonstrated in the subcutaneous tissues, in the tendons, periarticular structures, in muscle, about the sweat glands and in perineural structures. It is no wonder then that the extremity in cases of late trench foot tends to be stiff, painful on weight bearing and swollen after exercise even when its clinical appearance is quite normal.

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